We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800
Open access books available

122,000
International authors and editors

135M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS BOOK CITATION INDEX INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Chapter 4

Spinal Dural Arteriovenous Fistulas — Treatable Cause of Myelopathy

Antoine Nachanakian, Antonios El Helou,
Ghassan Abou Chedid and Moussa Alaywan

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/56725

1. Introduction

Arteriovenous fistula is an abnormal connection between a feeding artery and an adjacent vein. It can be congenital, acquired or created (e.g., brachial arteriovenous fistula for haemodialysis access). When it is pathological, its symptoms and diagnosis differ from one site to another in the human body.

Spinal vascular malformations (SVM) are rare diseases in comparison to vascular malformations of the brain. Spinal dural arteriovenous fistulas (SDAVFs), also known as Type I spinal arteriovenous malformations, account for 70% of spinal vascular malformations, and are the most common vascular malformations of the spinal cord and its surrounding dura mater; however, they remain relatively under-diagnosed. The majority of SDAVFs occur spontaneously, but a post-traumatic aetiology cannot be excluded in a significant proportion of them (Aghakhani N et al., 2008).

Hebold and Gaupp are credited with the first descriptions of isolated spinal cord vascular malformation in 1885 and 1888, respectively.

The first detailed clinical and pathological description of what is most likely to represent SDAVF is found in the 1926 report by Foix and Alajouanine.

Only recently did Kendall and Logue report the first case of an SDAVF as a distinct entity. Merland et al.15 illustrated the anatomical and pathological structure and location of an SDVAF in the dura mater and the intradural site of a radicular pouch. Finally, McCutcheon et al. worked on the anatomical characteristics of SDAVFs.

Diagnosis of SDAVFs is very difficult in clinical practice. In fact, symptoms are generally nonspecific.

Typically, this disease affects male patients in their 50s and 60s, causing progressive weakness of the lower limbs. Only 1% of patients are younger than 30 years of age. Most SDAVFs are solitary lesions, located between T6 and L2. Together, cervical and sacral SDAVF constitute fewer than 6% of cases. Multiple lesions in the same patient can exist, but this is rare.

2. Classification of SDAVF

According to the Spetzler classification, spinal vascular lesions can be subdivided into neoplasms, aneurysms and arteriovenous lesions. Arteriovenous lesions are further classified as arteriovenous fistulas and AVMs (Table 1).

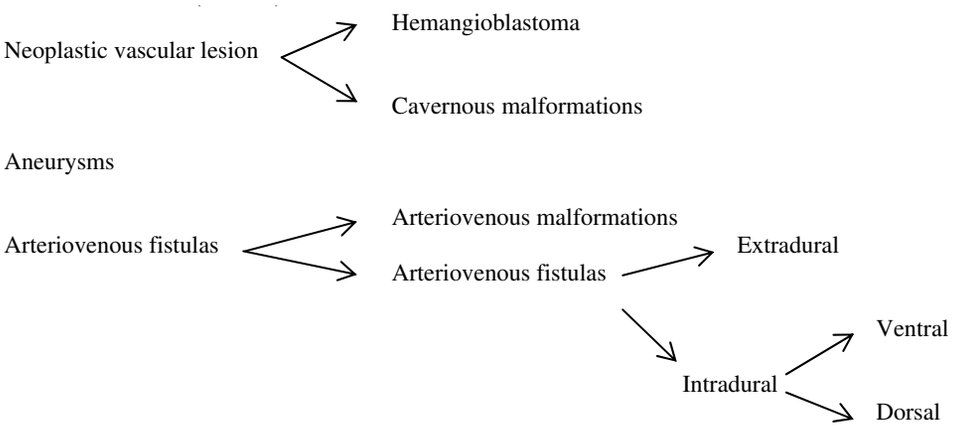

Table 1. Spetzler Classification of Spinal Vascular Malformation

Arteriovenous fistulas can either be extradural or intradural. Extradural arteriovenous fistulas have also been called epidural fistulas, and consist of a shunt between an extradural artery and a vein. Myelopathy may occasionally develop because of vascular steal (Arnaud et al., 1994a; Goyal et al., 1999).

Intradural arteriovenous fistulas are lesions causing progressive myelopathy. They may be located ventrally or dorsally. In dorsal intradural arteriovenous fistulas (SDAVF, Type I), the abnormal connection is formed between an artery and a vein at the level of a dural root sleeve with low flow, in contrast to the high-flow ventral type of fistulas. Ventral intradural arteriovenous fistulas consist of a shunt between the anterior spinal artery and an enlarged venous draining system (Djindjian et al., 1977). Type I fistulas are fed by radicular arteries and are low-pressure and low-flow vascular shunts. They are located within the dural sleeve of a nerve root and rarely entail haemorrhage; they are more commonly associated with myelopathy. Type II fistulas are located within the spinal cord itself and are true pial AVMs. They are high-pressure and high-flow shunts that are typically supplied by feeders off the anterior or

http://dx.doi.org/10.5772/56725

posterior spinal artery or the vertebral artery. These fistulas can be associated with myelopathy or haemorrhage. Type III SDAVFs are also high-flow shunts, derived from any feeding arteries but extending both intra- and extra-durally. They are extremely rare, but also difficult to treat because of their size. Type IV SDAVFs are perimedullary fistulas, with variable flow and pressure, which can be associated with myelopathy or haemorrhage (Table 2).

Type	Characteristics	Flow
I	Direct fistula in dural sleeve of nerve root	Low pressure, low flow
II	Intramedullary	High flow, high pressure
III	Intra & extramedullary paraspinal juvenile	High pressure
IV	Intradural extramedullary direct fistula	Low to high pressure Medium to high flow

Table 2. Classification of Arteriovenous fistula.

3. Evolution from embryology to pathology

3.1. Embryology of spinal vasculature

Embryological development of the spinal vasculature occurs in four stages. The primitive segmental stage starts in the second week of gestation. 31 pairs of segmental vessels originate from paired dorsal aortas and move towards the neural tube along the developing nerve roots. Segmental vessels divide into ventral and dorsal branches and form capillary networks on the ventrolateral surface of the neural tube. These networks develop into paired primitive ventral arterial tracts, precursors of an anterior spinal artery (K. Jellema et al., 2006).

The initial stage starts in the third week. At this level, dorsal arteries anastomose with the longitudinal venous channel dorsally and ventrally.

The transitional stage follows the initial stage in the sixth week of gestation. At this stage, fusion of ventral long arterial tracts and a decrease in segmental arteries occur. At ten weeks we have the adult pattern of spinal vasculature.

Finally, the terminal stage starts after the fourth month. This stage is characterized by the maturation and increase of the tortuosity of major spinal vessels.

3.2. Anatomy of spinal vasculature

The radiculomeningeal arteries are branches of the segmental arteries (thoracic intercostal in the thoracic spine, lumbar arteries in the lumbar spine and branches of the vertebral, the deep cervical and ascending cervical arteries in the cervical spine), supplying the dura in the spinal

canal, and are found at almost every spinal level. They should be distinguished from the radiculomedullary arteries, which exist only at some levels and supply the anterior and posterior spinal arteries that perfuse the spinal cord.

The venous drainage of the spinal cord does not parallel the arterial supply like the extrinsic arterial system; the extrinsic venous system has two parts: (a) longitudinal trunks, and (b) a pial plexus, termed the coronal venous plexus. The main arterial supply, the anterior spinal artery, is on the anterior surface of the cord, whereas the major venous channel is on the posterior surface, the posterior median vein. This longitudinal trunk vein has a midline location, whereas the relatively small posterior arterial trunks are off midline. In the thoracolumbar region, there is usually just one large anterior and one or two large posterior medullary veins, measuring up to 1.5 mm, accompanying lower thoracic or upper lumbar roots (Tadie M et al., 1985).

3.3. Pathophysiology of SDAVF

The onset in middle age suggests that SDAVF is an acquired condition, in contrast to intradural ventral fistulas or AVMs, which are assumed to be congenital abnormalities (Rosenblum et al., 1987). There are several other differences between SDAVF and AVMs. An SDAVF is never located within the spinal parenchyma. Patients with SDAVF very rarely suffer spinal haemorrhage. There are no associated vascular lesions in SDAVF. Intradural AVMs occur much more often in the cervical region (Rosenblum et al., 1987).

Aminoff and others proposed in 1974 that venous hypertension, rather than vascular steal, cord compression or haemorrhage, was the main pathophysiological factor (Aminoff et al., 1974). The shunt is most often formed within the dorsal surface of the dural root sleeve in the intervertebral foramen, where the radicular vein pierces the dura, together with one or more dural branches of the radicular artery. However, the shunt is sometimes situated along the dura between two adjacent nerve roots (Berenstein et al., 2004).

The increased pressure causes the venous system to 'arterialize', that is, the walls of intramedullary veins become thickened and tortuous. The radicular feeding artery is often a dural branch and, in a minority of cases, the medullary artery.

An increase in arterial pressure during the operation directly leads to an increase in venous pressure (Hassler et al., 1989), which may explain why some patients report that symptoms become worse after physical activity (Aminoff and Logue, 1974a; Khurana et al., 2002). Apart from the increased pressure caused by the shunt, the venous outflow may be less efficient to start with than is the case in healthy individuals (Merland et al., 1980; Thron, 2001).

The lower thoracic region has relatively fewer venous outflow channels at a segmental level than the cervical or lumbosacral region (Tadie et al., 1985). These differences in segmental outflow probably contribute to the phenomenon whereby venous congestion is transmitted in a caudo-cranial direction throughout the spinal cord, and to the fact that the first symptoms of myelopathy tend to reflect dysfunction of the lowest part of the cord, that is, the conus medullaris, even though the shunt is at the thoracic level. Possibly arteriovenous shunts are

http://dx.doi.org/10.5772/56725

not uncommon but they become symptomatic only through congenital or environmental factors that lead to impairment of venous outflow.

The typical pathophysiologic mechanism of SDAVF is spinal cord venous hypertension, which is caused by the presence of one or a few small low-flow arteriovenous shunts between a radiculomeningeal artery and a radiculomedullary vein, usually located in the intervertebral foramen within the dura.

Thus, SDAVFs are supplied by meningeal branches that do not perfuse the spinal cord, excluding arterial steal as an associated mechanism. The SDAVF drains via a radiculomedullary vein (almost always dorsal to the cord) into the perimedullary venous system, ultimately coalescing with normal spinal cord venous drainage in a retrograde fashion. The venous drainage of the DAVF is slow and extensive along the spinal veins, and may reach the cervical spinal canal and the cranial fossa in an ascending fashion and the veins of the cauda equina in a descending fashion (Patsalides et al., 2011).

The radiculomedullary veins draining the spinal cord venous flow to the epidural space are not anatomically numerous, and SDAVF is often associated with thrombosis of radiculomedullary and epidural veins. That explains why in SDAVF a low-flow arteriovenous shunt induces high venous pressure while in congenital high-flow arteriovenous malformations venous hypertension has fewer physiopathological consequences. The pressure in the vein draining the SDAVF rises to two thirds of the mean arterial pressure, resulting in venous hypertension, which then leads to decreased arteriovenous gradient and decreased venous drainage of the spinal cord parenchyma.

Due to the slow-flow characteristics of SDAVFs, haemorrhage rarely occurs. Even though the pathophysiology of SDAVFs located on the cervical dura is similar to DAVFs in the thoracolumbar area, they may cause spinal subarachnoid haemorrhage or intracranial haemorrhage if there is venous reflux towards the brain.

3.4. Clinical presentation

Symptoms consist of myelopathy and radiculopathy and can mimic a polyradiculopathy or anterior horn cell disorder. Symptoms can progress to paraparesis or quadriparesis.

At first, almost all patients report back or leg pain with mild sensory dysfunction. Then symptoms progress slowly until diagnosis (Diaz R J et al., 2008). The mean time from the initial onset of symptoms is 15 to 23 months. In general, upper motor neuron lesion manifestations are dominant. Progressive weakness, muscle spasms, faecal incontinence, overflow urinary incontinence or urinary retention and erectile dysfunction are characteristic of myelopathy, but they are not specific to spinal dural arteriovenous fistula (Jellema K et al., 2006).

3.5. Deferential diagnosis

The differential diagnosis of nontraumatic progressive myelopathy is broad (K. Jellema, et al., 2006). The most urgent diagnoses to exclude are compressive neoplasm and infection with a spinal epidural abscess. However, other causes of increased MRI T2 cord signal are intrame-

dullary tumours, degenerative disc disease, inflammatory and autoimmune conditions, infections, vascular disorders, and nutritional and toxic causes. Intradural tumours can be primary or metastatic and are found in the intramedullary or extramedullary space. Myelopathy due to cervical spondylosis is the most common cause of nontraumatic spastic paraparesis and quadriparesis. Lumbar canal stenosis is also common in this age group and can contribute to gait dysfunction, thus complicating the diagnosis. Myelitis can be acute (as seen in postviral infection or demyelinating myelitis) but can also be subacute to chronic conditions (e.g., AIDS myelopathy, syphilis). Inflammation of the spinal cord also occurs in several rheumatologic and connective tissue diseases, which may precede the onset of systemic symptoms by years. 16 Nutritional deficiencies should be considered in patients with gastrointestinal disease, a history of gastric bypass surgery, or a history of exposures to toxins that prevent adequate absorption of nutrients.

The time course, patient age, comorbidities, systemic symptoms, presence or absence of peripheral nervous system involvement, and localization to tracts or regions within the spinal cord can help narrow the extensive differential diagnosis for progressive myelopathy.

Many aetiologies are easily excluded by history, imaging (e.g., tumours), and serum and cerebrospinal fluid analysis (e.g., infections). However, the nonspecific features of SDAVF and frequency of both upper motor neuron signs (increased muscle tone and deep tendon reflexes) and lower motor neuron signs (flaccid weakness, depressed deep tendon reflexes) can delay diagnosis, particularly in older adults who are likely to have comorbid systemic diseases and/or cervical spondylosis. Failure to respond to standard therapy for other causes of myelopathy should trigger further investigation.

3.6. Imaging

The essential investigations to establish the diagnosis are MRI and catheter angiography, which should be performed when a progressive myelopathy is suspected. MRI findings include hypo-intensities on T1-weighted images and hyperintensities on T2-weighted images. Increased signal intensity in the centre of the spinal cord and peripheral sparing on T2-weighted images is found in 67–100% of patients (Figure. 1) (Bowen et al., 1995; Hurst and Grossman, 2000; Luetmer et al., 2005).

In addition, abnormalities suggesting abnormal blood vessels may be seen on either the ventral or the dorsal side of the spinal cord. These 'flow void phenomena' representing tortuous and dilated veins at the dorsal surface of the spinal cord are found in 35–91% of patients (Hurst and Grossman, 2000). It seems that the flow voids are found more often, as studies are more recent, which may reflect advancement in MR techniques (Hurst and Grossman, 2000). The central hyperintense lesions are sometimes difficult to interpret, and may resemble anterior spinal artery infarction, myelitis or spinal cord neoplasms (Grandin et al., 1997), or, if slit-like, a persistent central canal (Holly and Batzdorf, 2002). Gadolinium-enhanced MRI scanning may reveal some contrast enhancement of the spinal cord (Terwey et al., 1989).

http://dx.doi.org/10.5772/56725

Figure 1. Sagittal T2-weighted images showing intramedullary hyperintensity.

MR angiography reveals flow in serpentine perimedullary structures (Bowen et al., 1995; Binkert et al., 1999; Mascalchi et al., 2001). MR angiography may also give an indication about the level of the SDAVF, which helps to confine the extent and duration of catheter angiography (Bowen et al., 1995; Mascalchi et al., 1999; Luetmer et al., 2005).

On the other hand, false positive MR angiography is also possible, in that normal vessels may be interpreted as being pathologically enlarged (Binkert et al., 1999; Luetmer et al., 2005).

MR TRICKS is a better tool that has improved the diagnosis of SDAVF and reduced the false positive of MR angiography (Korosec et al., 1996). First introduced by the University of Wisconsin-Madison group, time resolved imaging of contrast kinetics (TRICKS) is a method of 3D contrast-enhanced magnetic resonance angiography (MRA) providing temporal information. TRICKS combines variable rate k-space sampling, temporal interpo-

lation of k-space views, view sharing and zero filling in the slice-encoding dimension. The interesting aspect of the method is its ability to acquire a pure arterial weighted phase; TRICKS allows ultraresolved 3D contrast-enhanced MRA without venous contamination (Albini Ricoli et al., 2007).

Before the introduction of MRI, diagnosis was often made by means of myelography (Gilbertson et al., 1995). This investigation would show an irregular, varicose dilation of the lumbar veins, sometimes giving the lumbar roots a 'postage stamp' appearance.

Catheter angiography is still the gold standard in the diagnosis of SDAVF (Figure 2). Not only the intercostal and lumbar arteries should be visualized as potential feeding arteries of an abnormal shunt, but also the median and lateral sacral artery, the deep cervical and ascending cervical arteries.

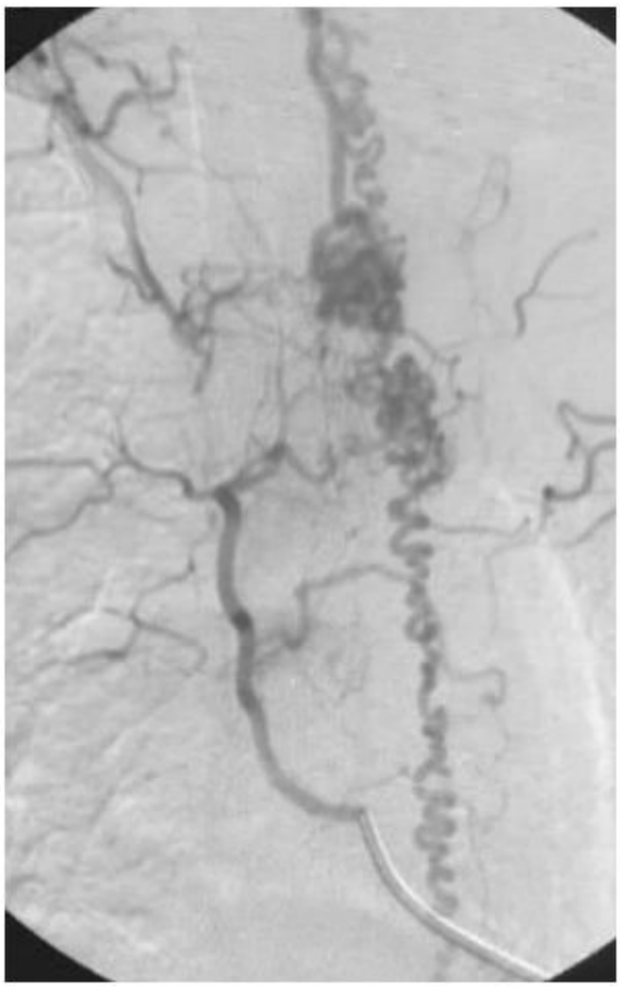

Figure 2. Spinal angiography illustrating a SDAVF fed by right Th11 radicular artery with tortuous draining veins.

The angioarchitecture of the fistula should be thoroughly investigated, especially with regard to the question of whether the arterial feeder is a dural branch or a segmental medullary artery, which also contributes to the anterior spinal artery. In the latter case, endovascular treatment

http://dx.doi.org/10.5772/56725

is not possible, because infarction of the spinal cord is likely to occur. Furthermore, it is essential to identify the artery of Adamkiewicz, because the fistula may originate from this important tributary to the anterior spinal artery.

4. Decision making and management

4.1. Diagnostic criteria

The typical patient where SDAVF is suspected is aged 40–80 and shows a gradual onset of slowly progressive or stepwise worsening myelopathy characterized by lower-extremity weakness, sensory loss to pinprick and light touch, and late development of bowel and bladder dysfunction. Pre-operative imaging is mandatory.

MR imaging is an initial diagnostic tool. MRI assesses the level of the suspected SDAVF, shown by the flow void and the tortuosity of the vessels in addition to intramedullary hyper signal on a T2-weighted image. MRA can play a valuable role in confirming the diagnosis and targeting conventional catheter angiography.

All patients with confirmed or suspected SDAVF are evaluated by spinal angiography (Figure 3). After confirming the diagnosis, a joint decision is taken by the neurosurgery team and endovascular neuroradiology about the method of treatment.

Figure 3. Spinal angiography of the left Th6 radicular artery showing SDAVF with tortuous veins extending superior to the cervico-thoracic junction level.

4.2. Indication for surgical treatment

Treatment for SDAVFs must be performed as soon as possible and may be surgical or endovascular, as both are safe and effective. Microsurgical treatment was considered the gold standard for many years but recent technical advances in endovascular surgery have made endovascular treatments an option. As with all inherent disease processes of the spinal cord, post-operative function is highly related to pre-operative presentation, and maximum functional results are obtained in patients treated early, before advanced deterioration has taken place.

4.3. Surgery

4.3.1. Pre-operative work up

All patients admitted to neurosurgery wards are clinically evaluated the day before the procedure. A full neurological examination is performed evaluating the motor, sensory, and deep tendon reflexes, as well as the gait. In addition, clinical urological evaluation and, when needed, urodynamic studies are carried out.

4.3.2. Operative procedure

The procedure is carried out under general anaesthesia. All patients are operated in a prone position, with the thoracolumbar spine segment in neutral to a slightly kyphotic position, avoiding hyperlordosis.

Intra-operative motor-evoked potential monitoring is used in all procedures with continuous observation by the neurologist until the end of the procedure.

The level of the procedure is localized after positioning under fluoroscopy. Midline vertical skin incision is carried out. The subcutaneous layer and the paraspinal muscles are dissected until the articular facets are identified.

A two-level laminectomy is carried out. A median longitudinal dural incision is performed, exposing the intradural nerve root, initial segments of draining vein and several millimetres of the feeding radicular artery (Figure 4). Intra-operative Doppler control is performed upon identification of the fistula revealing an arterial spectrum on the redundant dorsal medullary veins. After the clipping of the feeder of the arteriovenous shunt by a temporary clamp, the intra-operative monitoring documents a complete disappearance of the arterial spectrum and the reappearance of the venous pattern. In addition, the motor-evoked potential is monitored and controlled twice, at a five-minute interval. If no changes are observed in the physiological studies, the fistula is obliterated at the artery vein connection with two small permanent vascular clips. Then, it is coagulated (Figure 5).

Dural closure is performed with fascia-latta auto graft using a tight continuous running suture method. Some trials for synthetic dural graft fail due to a high rate of post-operative CSF leak.

http://dx.doi.org/10.5772/56725

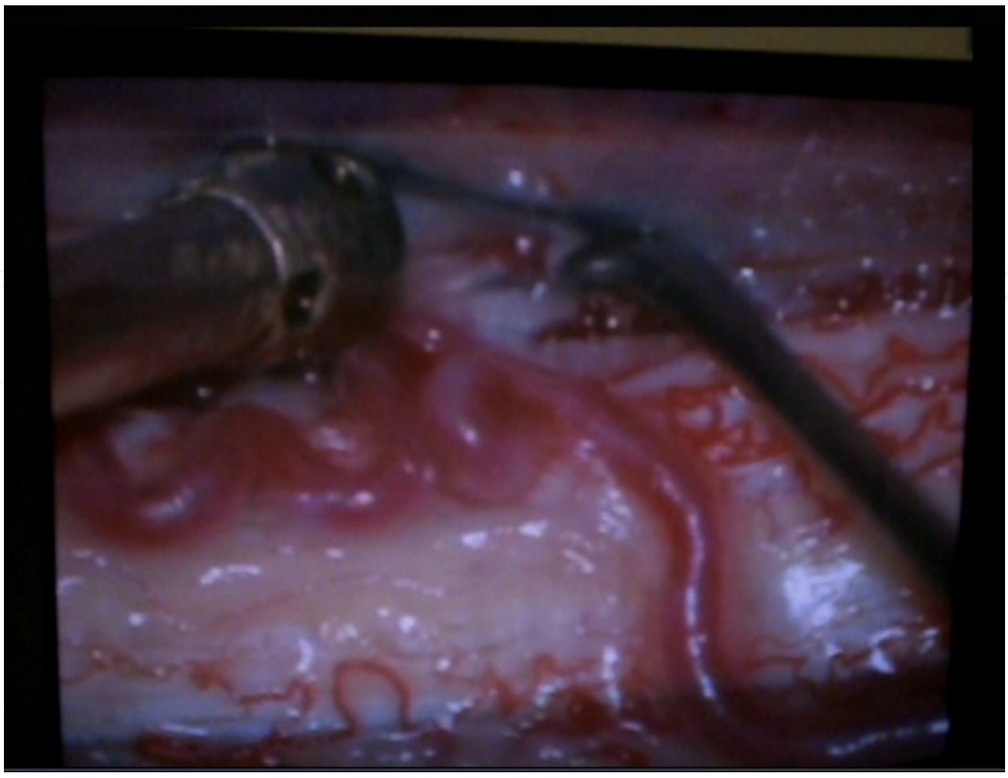

Figure 4. Pre-operative findings showing tortuous veins with artery under suction tip accompanying the nerve root beneath the spatula.

4.3.3. Post-operative care

The patient is kept in a completely supine position to avoid CSF leak and is out of bed 48 hours after surgery. Routine laboratory tests are carried out on day 1. Steroids are applied post-operatively in high dosages (Methylprednisolone, 1g every 24 hours; or Dexamethasone, 8mg every 8 hours), and this regime is continued with progressive tapering over two weeks, with a shift to oral medication in 72 hours. No imaging is ordered in the immediate post-op period, unless there is a clinical deterioration. A control spine MRI with angiography MR is carried out six months after the operation.

4.3.4. Long-term results

Overall improvement was noted in surgically treated patients, and to a considerable degree of satisfaction in approximately 75% of patients. Motor strength and gait improvement were superior to bladder function (Ropper A E et al., 2012). Deterioration of the previous neurological status was found in less than 10% of cases; persistence of stable status was found in 10–15% of the operated patients (Schick U et al., 2003, Song JK et al., 2001).

Post-operative neurological improvement progressively increased during the next three months. Patients achieved maximum improvement after an average period of six months. The prominent characteristic of this surgery is a low level of post-operative pain.

Motor and gait dysfunction improved more than sensory and bladder dysfunction with time.

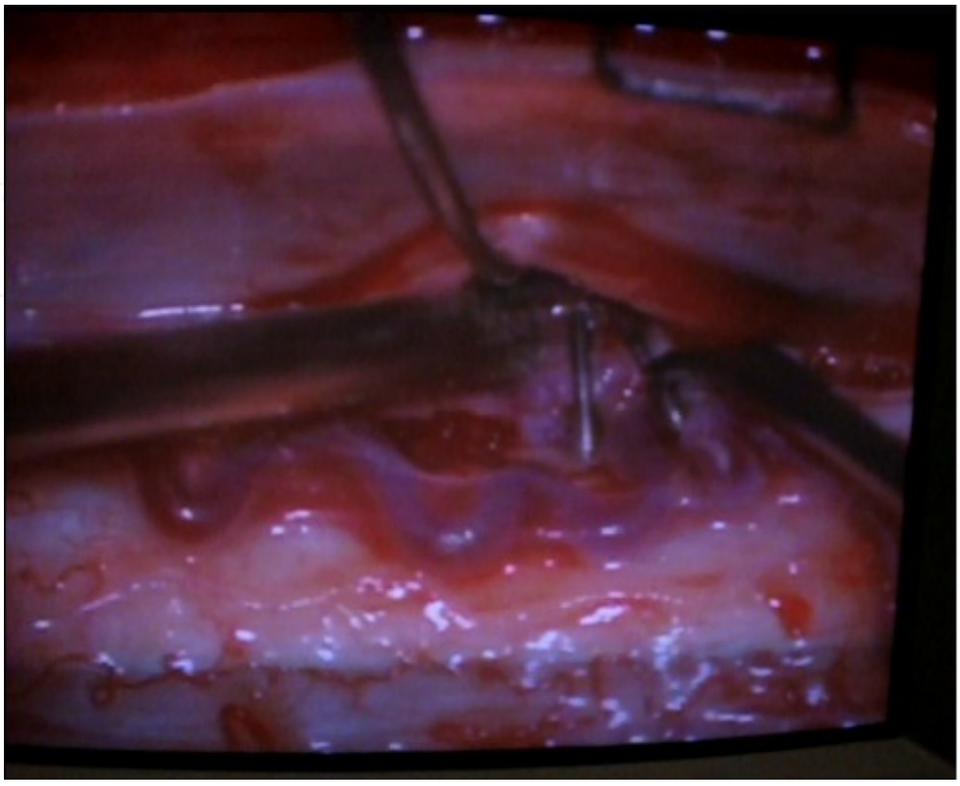

Figure 5. Pre-operative finding after clipping of the feeding artery. The image shows early shrinkage of the arterialized veins.

4.3.5. Complications

In general, this procedure is well tolerated. The overall rate of complications is between 1 and 5%. Two kinds of complications exist: the early and the delayed (Schick U et al., 2003, Song JK et al., 2001 and Ropper A E et al., 2012).

Early complications include CSF leak, infection, haematoma, and neurological sequelae.

Delayed complications depend on obliteration rate, which is around 98% in surgical patients.

4.4. Endovascular treatment

Endovascular therapy is less invasive than microsurgery, and allows both diagnosis and treatment in a single session (Park S B et al., 2008); however, more than one session may be necessary for some patients.

The ability to definitively treat spinal DAVFs using endovascular embolization has significantly improved over the last several decades. Overall rates of definitive embolization of spinal DAVFs have ranged between 25 and 100%, depending in part on the embolic agent used and the use of variable stiffness microcatheters. The majority of recent studies in which N-butyl

http://dx.doi.org/10.5772/56725

cyanoacrylate or other liquid embolic agents were used have reported success rates of 70–90% (Sivakumar W et al., 2009). Although endovascular therapy is potentially less invasive and associated with less morbidity and earlier mobilization than surgery, endovascular therapy has been associated with a lower initial success rate and higher rate of recurrence than microsurgical therapy (Van Dijk JM et al., 2002). Several factors need to be considered when selecting endovascular therapy for the treatment of spinal DAVF. A spinal DAVF usually consists of multiple dural arterial vessels with a single draining vein. Thus, occlusion of a feeding arterial vessel may lead to recanalization or collateral development in the early post-operative period (McCutcheon IE et al., 1996). Another important consideration is the identification of patients with conditions that would make them unsuitable for endovascular therapy. Embolization therapy may not be feasible if the arterial feeder is too small to catheterize and arterial damage due to catheter manipulation is likely, as in patients with severe arteriosclerosis, or if the anterior spinal artery, the Adamkiewicz and feeding artery of the fistula originate from the same segmental artery (Thron A, 2001).

Detachable coils, silk sutures, polyvinyl alcohol (PVA), and n-butyl-2-cyanoacrylate (NBCA) have been used as embolic agents for endovascular management of DAVFs.

The use of liquid embolization material is imperative to prevent recanalization, while the use of particle embolization (polyvinyl alcohol, embospheres and gel foam) is not indicated because of its high recanalization rates. At first, N-butyl cyanoacrylate (NBCA) was often used.

Endovascular embolization of SDAVF using NBCA has a high success rate (Warakaulle DR et al., 2003). It is referred to as glue, being an extremely effective liquid acrylic polymer that polymerizes when it comes into contact with an ionic medium such as blood. NBCA is diluted with ethiodized oil to render it radiopaque and visible during the embolization. Transarterial *NBCA* embolization is highly operator-dependent because the glue quickly polymerizes, often leading to inadequate filling of the proximal draining vein. Complete filling of the proximal vein is a critical component for definitive endovascular cure of these lesions.

At present, the Onyx is used, which is a new liquid embolic agent, a mixture of ethylene-vinyl alcohol copolymer and dimethyl sulfoxide (DMSO). While DMSO diffuses under aqueous conditions the ethylene-vinyl alcohol copolymer precipitates and mechanically occludes the feeding vessels, whose viscosity makes it suitable for treatment of spinal DAVF, where penetration into the proximal radicular vein is required (Carlson AP et al., 2007; Ross et al., 2012). Contrary to NBCA, Onyx does not adhere to the biological surfaces, which allows for slow delivery and improved penetration of the nidus (Nogueira RG et al., 2009).

4.4.1. Endovascular technique

The procedure is carried out under general anaesthesia in an interventional radiology room. The patient is in a supine position. The right femoral crease is prepared for puncture.

Neurophysiological monitoring allows a warning for the neuroradiologist of impending irreversible neurological damage so that action may be taken for the prompt restoration of adequate spinal cord perfusion. Muscle motor-evoked potentials reflect spinal cord perfusion

in the anterior spinal artery territory better than somatosensory-evoked potentials (SEPs). Although rarely used, motor-evoked potentials make the procedure safer (Sala F et al., 2001).

After recording baseline SEPs and MEPs, 50 mg of sodium amytal is injected through the microcatheter at the position of the intended embolization, followed by assessment of SEPs and MEPs. If no changes have occurred, 40 mg of lidocaine is then injected, followed by recording of SEPs and MEPs. If no changes are noted again, embolization is performed (Niimi et al., 2000). If there is any change in either the SEPs or the MEPs, NBCA embolization is not performed from that catheter position.

After delivery of the embolization material, DMSO leaks from the embolus allowing for Onyx to precipitate into the vessel lumen. (Figure 6).

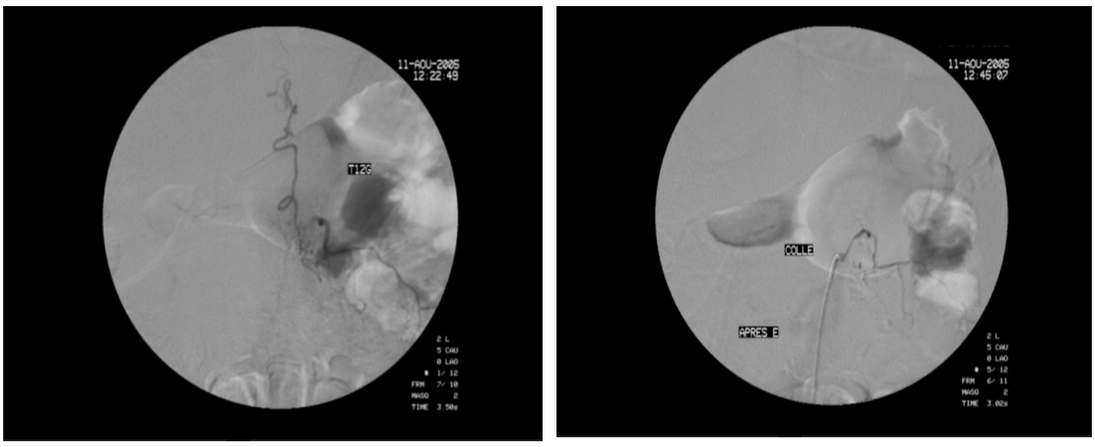

Figure 6. Left: Arteriography of SDAVF pre embolization. Right: Arteriography of the same patient post embolization.

4.4.2. Post-procedure care

The patient is transferred to a regular ward after the embolization procedure. The post-operative steroid protocol is identical to the one used in the microsurgery technique (high-dose steroids for the first two days, then progressive tapering after a shift to per os intake over two weeks).

The patients ambulate on the first day post-embolization and can be discharged on day 2. Regular clinical follow-ups are carried out one, three and six months after the procedure. A new spinal angiography is ordered six months after the endovascular procedure.

4.4.3. Complications

The risk of inadvertent embolization of arteries feeding normal tissue is high, and precise positioning of the delivery microcatheter as close as possible to the arteriovenous lesion is an important manoeuvre to prevent embolization of normal tissue. To prevent polymerization in the delivery catheter or prematurely in the vasculature, the microcatheter must first be thoroughly flushed. For NBCA solution after mixing with radiopaque solution, the micro-

http://dx.doi.org/10.5772/56725

catheter is flushed with 10–15 ml of 5% dextrose solution. On the other hand, the use of Onyx injection necessitates microcatheter flushing with saline solution to fill the dead space estimated for 0.5 to 1 ml.

The recanalization rate in all serious cases is 15–20% (Song et al., 2001; Hall WA et al., 1989). The bleeding rate and/or embolization of inadequate arteries differed between serious and other cases, fluctuating between 0 and 4% (Schick U and Hassler W, 2003; Steinmetz MP et al. 2004).

5. Institution experience

Our experience is based on 16 cases of SDAVF treated from January 2003 to March 2012. Eleven cases were managed surgically, whereas five were treated by endovascular embolization. This decision was based on age, clinical symptoms, and the level of the fistula.

87.5% of our patients were male. The mean age was 65 years. Three cases were in the lumbar level, one in the sacral and 12 in the thoracic region. Fifteen cases had a radicular artery feeder, one had an intercostal feeder and one was identified incidentally during an interlaminar approach for a herniated disc.

We recorded three cases of radiculopathy in our series. All other patients showing signs of progressive myelopathy worsened over long-term periods.

In our institution, patients are admitted for 24 hours before the day of the procedure, and as early as possible after initial diagnosis.

In the pre-operative period, the patient is treated with anti-platelets and other anti-coagulation agents for five days. Only LMWH can be tolerated up to 12 hours before the procedure.

If the treatment decided upon is surgical, patients ambulate 48 hours after the procedure; those treated by endovascular embolization ambulate within 24 hours of the procedure.

All patients are assessed using the Aminoff-Logue disability scale in the pre-op and post-op periods.

Usually, patients have regular follow-ups at one, three and 12 months.

Patients at first reported an improvement of their radicular pain, with a mean reduction of 4.3/10 on visual analogue scale (VAS) (0: absent pain, to 10: severe intolerable pain necessitating intra-venous treatment). In the pre-operative period, radicular pain when extant had a mean score of 7.8/10 on VAS; in the immediate post-op period, the pain was 3.4/10 on VAS.

We did not observe any complications in the surgically treated patients, although recanalization occurred in one of the patients treated by embolization. No bleeding or embolization at other sites was observed.

6. Conclusion

With progressive myelopathy of unknown origin in a middle-aged patient, SDAVF must always be considered. Magnetic resonance imaging, especially the new 3T generation, is a good initial tool to identify the tortuous veins around the spinal cord and spinal cord oedema, especially on T2WI. Spinal catheter angiography remains the gold standard for diagnosis and identification of the site of the fistula. These vascular malformations can be treated either by endovascular embolization or surgical clipping, keeping in mind that the latter has a higher occlusion rate.

Author details

Antoine Nachanakian, Antonios El Helou, Ghassan Abou Chedid and Moussa Alaywan

Division of Neurosurgery and Endovascular Neuroradiology, Saint George Hospital University Medical Center, Balamand University, Lebanon

References

[1] Aghakhani N, Parker F, David P, et al. Curable cause of paraplegia: spinal dural arteriovenous fistulae. Stroke 2008; 39: 2756e9.

[2] Albini Riccioli L, Marliani A F, Ghedin P, Agati R, and Leonardi M. CE-MR Angiography at 3.0 T Magnetic Field in the Study of Spinal Dural Arteriovenous Fistula. Interv Neuroradiol 2007; 13(1): 13–18.

[3] Aminoff MJ, Barnard RO, Logue V. The pathophysiology of spinal vascular malformations. J Neurol Sci 1974; 23: 255–63.

[4] Arnaud O, Pelletier J, Dalecky A, Cherif AA, Azulay JP, Salamon G, et al. Spinal dural fistula with peri-medullar venous drainage. Rev Neurol (Paris) 1994b; 150: 713–20.

[5] Patsalides A, Santillan A, Knopman J, Tsiouris AJ, Riina HA, Gobin YP. Endovascular management of spinal dural arteriovenous fistulas. J NeuroIntervent Surg 2011; 3: 80e84. doi:10.1136/jnis.2010.003178.

[6] Berenstein A, Lasjaunias P, Ter Brugge KG. editors. Surgical neuroangiography 2.2. Berlin: Springer; 2004.

[7] Binkert CA, Kollias SS, Valavanis A. Spinal cord vascular disease: characterization with fast three-dimensional contrast-enhanced MR angiography. AJNR Am J Neuroradiol 1999; 20: 1785–93.

[8] Bowen BC, Fraser K, Kochan JP, Pattany PM, Green BA, Quencer RM. Spinal dural arteriovenous fistulas: evaluation with MR angiography. AJNR Am J Neuroradiol 1995; 16: 2029.

[9] Carlson AP, Taylor CL, Yonas H. Treatment of dural arteriovenous fistula using ethylene vinyl alcohol (onyx) arterial embolization as the primary modality: Short-term results. J Neurosurg 2007; 107: 1120-5.

[10] Diaz RJ and Wong JH. Spinal dural arteriovenous fistula: a treatable cause of myelopathy. CMAJ 2008; 178(10): 1286–1288.

[11] Djindjian M, Djindjian R, Rey A, Hurth M, Houdart R. Intradural extramedullary spinal arterio-venous malformations fed by the anterior spinal artery. Surg Neurol 1977; 8: 85–93.

[12] Eskandar EN, Borges LF, Budzik RF Jr, et al. Spinal dural arteriovenous fistulas: experience with endovascular and surgical therapy. J Neurosurg 2002; 96(2 Suppl): 162e7.

[13] Gilbertson JR, Miller GM, Goldman MS, Marsh WR. Spinal dural arteriovenous fistulas: MR and myelographic findings. AJNR Am J Neuroradiol 1995; 16: 2049–57.

[14] Goyal M, Willinsky R, Montanera W, Ter Brugge K. Paravertebral arteriovenous malformations with epidural drainage: clinical spectrum, imaging features, and results of treatment. AJNR Am J Neuroradiol 1999; 20: 749–55.

[15] Grandin C, Duprez T, Stroobandt G, Laterre EC, Mathurin P. Spinal dural arterio-venous fistula: an underdiagnosed disease? Acta Neurol Belg 1997; 97: 17–21.

[16] Guo LM, Zhou HY, Xu JW, et al. Dural arteriovenous fistula at the foramen magnum presenting with subarachnoid hemorrhage: case reports and literature review. Eur J Neurol 2010; 17:684e91.

[17] Hall WA, Oldfield EH, Doppman JL. Recanalization of spinal arteriovenous malformations following embolization. J Neurosurg. 1989; 70:714–720.

[18] Hassler W, Thron A. Flow velocity and pressure measurements in spinal dural arteriovenous fistulas. Neurosurg Rev 1994; 17: 29–36.

[19] Hurst RW, Grossman RI. Peripheral spinal cord hypointensity on T2-weighted MR images: a reliable imaging sign of venous hypertensive myelopathy. AJNR Am J Neuroradiol 2000; 21: 781–6.

[20] Holly LT, Batzdorf U. Slitlike syrinx cavities: a persistent central canal. J Neurosurg 2002; 97: 161–5.

[21] Jellema K, Sluzewski M, van Rooij WJ, et al. Embolization of spinal dural arteriovenous fistulas: importance of occlusion of the draining vein. J Neurosurg Spine 2005; 2: 580e3.

[22] Jellema K, Tijssen CC, van Gijn J. Spinal dural arteriovenous fistulas: a congestive myelopathy that initially mimics a peripheral nerve disorder. Brain 2006; 129: 3150–64.

[23] Khurana VG, Perez-Terzic CM, Petersen RC, Krauss WE. Singing paraplegia: a distinctive manifestation of a spinal dural arteriovenous fistula. Neurology 2002; 58: 1279–81.

[24] Korosec FR, Frayne R, et al. Time-resolved contrast-enhanced 3D MR angiography. Magn Reson Med. 1996; 36: 345–351.

[25] Luetmer PH, Lane JI, Gilbertson JR, Bernstein MA, Huston J III, Atkinson JL. Preangiographic evaluation of spinal dural arteriovenous fistulas with elliptic centric contrast-enhanced MR angiography and effect on radiation

[26] dose and volume of iodinated contrast material. AJNR Am J Neuroradiol 2005; 26: 711–8.

[27] Mascalchi M, Ferrito G, Quilici N, Mangiafico S, Cosottini M, Cellerini M, et al. Spinal vascular malformations: MR angiography after treatment. Radiology 2001; 219: 346–53.

[28] Merland JJ, Riche MC, Chiras J. Intraspinal extramedullary arteriovenous fistulae draining into the medullary veins. J Neuroradiol 1980; 7: 271e320.

[29] Merland JJ, Assouline E, Rufenacht D, Guimaraens L, Laurent A. Dural spinal arteriovenous fistulae draining into medullary veins: clinical and radiological results of treatment (embolization and surgery) in 56 cases. Excerpta Med Int Congr Ser 1986; 698: 283–9.

[30] McCutcheon IE, Doppman JL, Oldfield EH. Microvascular anatomy of dural arteriovenous abnormalities of the spine: a microangiographic study. J Neurosurg 1996; 84: 215–220.

[31] Niimi Y, Sala F, Deletis V, Berenstein A. Provocative Testing for Embolization of Spinal Cord AVMs. Interv Neuroradiol. 2000; 30; 6 Suppl 1: 191–4.

[32] Nogueira RG, Dabus G, Rabinov JD, Ogilvy CS, Hirsch JA, Pryor JC. Onyx embolization for the treatment of spinal dural arteriovenous fistulae: Initial experience with long-term follow-up: Technical case report. Neurosurgery 2009; 64: E197–8.

[33] Park SB, Han MH, Jahng TA, Kwon BJ, and Chung CK. Spinal Dural Arteriovenous Fistulas: Clinical Experience with Endovascular Treatment as a Primary Therapeutic Modality. J Korean Neurosurg Soc 2008; 44(6): 364–369.

[34] Ropper AE, Gross BA, Du R. Surgical Treatment of Type I Spinal Dural Arteriovenous Fistulas. Neurosurg Focus 2012; 32(5): e3

[35] Rosenblum B, Oldfield EH, Doppman JL, Di Chiro G. Spinal arteriovenous malformations: a comparison of dural arteriovenous fistulas and intradural AVMs in 81 patients. J Neurosurg 1987; 67: 795–802

[36] Ross C, Puffer RC, Daniels DJ, Kallmes DF, Cloft HJ, Lanzino G. Curative Onyx Embolization of Tentorial Dural Arteriovenous Fistulas. Neurosurg Focus. 2012; 32(5): e4

[37] Sala F, Niimi Y, Berenstein A, Deletis V. Neuroprotective role of neurophysiological monitoring during endovascular procedures in the spinal cord. Ann N Y Acad Sci 2001; 939: 126–36.

[38] Schaat TJ, Salzman KL, Stevens EA. Sacral origin of a spinal dural arteriovenous fistula: case report and review. Spine (Phila Pa 1976) 2002; 27: 893e7.

[39] Schick U and Hassler W. Treatment and outcome of spinal dural arteriovenous fistulas. Eur Spine J. 2003; 12(4): 350–355.

[40] Sivakumar W, Zada G, Yashar P, Giannotta SL, Teitelbaum G, Larsen DW. Endovascular management of spinal dural arteriovenous fistulas. A review. Neurosurg Focus 2009; 26(5): E15.

[41] Song JK, Vinuela F, Gobin YP, Duckwiler GR, Murayama Y, Kureshi I, Frazee JG, Martin NA. Surgical and endovascular treatment of spinal dural arteriovenous fistulas: long-term disability assessment and prognostic factors. J Neurosurg. 2001; 94(2 Suppl): 199–204.

[42] Steinmetz MP, Chow MM, Krishnaney AA, Andrews-Hinders D, Benzel EC, Masaryk TJ, Mayberg MR, Rasmussen PA. Outcome after the treatment of spinal dural arteriovenous fistulae: a contemporary single-institution series and meta-analysis. Neurosurgery 2004; 55(1): 77–87.

[43] Tadie M, Hemet J, Freger P, Clavier E, Creissard P. Morphological and functional anatomy of spinal cord veins. J Neuroradiol 1985; 12: 3–20.

[44] Thron A. Spinal dural arteriovenous fistulas. Radiologe 2001; 41: 955–60.

[45] Terwey B, Becker H, Thron AK, Vahldiek G. Gadolinium-DTPA enhanced MR imaging of spinal dural arteriovenous fistulas. J Comput Assist Tomogr 1989; 13: 30–7.

[46] Thron A. Spinal dural arteriovenous fistulas. Radiologe 2001; 41: 955–960.

[47] Tsai LK, Jeng JS, Liu HM, Wang HJ, Yip PK. Intracranial dural arteriovenous fistulas with or without cerebral sinus thrombosis: analysis of 69 patients. J Neurol Neurosurg Psychiatry 2004; 75: 1639–41.

[48] Van Dijk JM, Ter Brugge KG, Willinsky RA, Farb RI, Wallace MC. Multidisciplinary management of spinal dural arteriovenous fistulas: clinical presentation and long-term follow-up in 49 patients. Stroke 2002; 33: 1578–1583

[49] Warakaulle DR, Aviv RI, Niemann D, Molyneux AJ, Byrne JV, Teddy P. Embolisation of spinal dural arteriovenous fistulae with Onyx. Neuroradiology 2003; 45(2): 110–2.

IntechOpen